AMENDEMENTS TO THE CLAIMS

Claims 1-28 (Canceled)

29. (New) A transgenic mouse whose genome comprises a disruption in an endogenous murine CX2 gene, wherein where the disruption is homozygous, the transgenic mouse lacks production of functional CX2 protein, and exhibits, relative to a wild-type mouse, at least one of increased seizure susceptibility, increased glucose tolerance, increased ability to metabolize glucose, increased body weight, increased body length and increased body weight to body length ratio.

30. (New) The transgenic mouse of claim 29, wherein the increased seizure susceptibility is characterized by a decreased response threshold to metrazol, relative to a wild-type control mouse.

31. (New) The transgenic mouse of claim 29, wherein the increased glucose tolerance or increased ability to metabolize glucose is characterized by a decrease in blood glucose level after administration of glucose, relative to a wild-type mouse.

32. (New) A cell or tissue obtained from the transgenic mouse of claim 29.

33. (New) A transgenic mouse comprising a heterozygous disruption in an endogenous murine CX2 gene, wherein the disruption in a homozygous state inhibits production of functional CX2 protein resulting in a transgenic mouse exhibiting, relative to a wild-type mouse, at least one of increased seizure susceptibility, increased glucose tolerance, increased ability to metabolize glucose, increased body weight, increased body length and increased body weight to body length ratio.

34. (New) The transgenic mouse of claim 33, wherein the increased seizure susceptibility is characterized by a decreased response threshold to metrazol, relative to a wild-type control mouse.

35. (New) The transgenic mouse of claim 33, wherein the increased glucose tolerance or increased ability to metabolize glucose is characterized by a decrease in blood glucose level after administration of glucose, relative to a wild-type mouse.

36. (New) A method of producing a transgenic mouse comprising a disruption in an endogenous murine CX2 gene, the method comprising:

    (a) introducing a targeting construct capable of disrupting the endogenous murine CX2 gene into a murine embryonic stem cell;

(b) introducing the murine embryonic stem cell into a mouse blastocyst;

(c) implanting the resulting blastocyst into a pseudopregnant mouse, wherein the pseudopregnant mouse gives birth to a chimeric mouse; and

(d) breeding the chimeric mouse to produce the transgenic mouse,

wherein where the disruption is homozygous, the transgenic mouse lacks production of functional CX2 protein and exhibits, relative to a wild-type mouse, at least one of increased seizure susceptibility, increased glucose tolerance, increased ability to metabolize glucose, increased body weight, increased body length and increased body weight to body length ratio.

37. (New) The transgenic mouse produced by the method of claim 36.

38. (New) A targeting construct comprising:

(a) a first polynucleotide sequence homologous to at least a first portion of an endogenous murine CX2 gene;

(b) a second polynucleotide sequence homologous to at least a second portion of the endogenous murine CX2 gene; and

(c) a selectable marker gene located between the first and second polynucleotide sequences.

39. (New) A method of producing a targeting construct, the method comprising:

(a) providing a first polynucleotide sequence homologous to at least a first portion of an endogenous murine CX2 gene;

(b) providing a second polynucleotide sequence homologous to at least a second portion of the endogenous murine CX2 gene;

(c) providing a selectable marker gene; and

(d) inserting the first sequence, second sequence, and selectable marker gene into a vector such that the selectable marker gene is located between the first and second sequences to produce the targeting construct.